

## HIV and AIDS – what was (again) not said on last year's World AIDS day

For World AIDS Day 2021 we would like to point out some publications that do not fit the picture of the all-round success story drawn of HIV and AIDS by the media and representatives of the pharmaceutical lobby.

The publications cited here are well known to virologists and molecular biologists. Many aspects are undisputed, but are not included in the reporting.

### A note on the context:

HIV is a virus of the lentivirus genus from the retrovirus family. These are viruses whose genetic information is stored as RNA and not, e.g. in humans, as DNA. HIV is said to be transmitted through blood or semen and counts as a sexually transmitted disease (STD). An infection with this virus is said to lead to a weakening of the immune system after 10-15 years, so that those affected begin to suffer from what are known as *opportunistic infections and diseases*. This is then called AIDS, *Acquired Immunodeficiency Syndrome*. The list of these approx. 30 classic diseases, also known as *AIDS-defining* diseases, has been expanded several times over the years and includes not only tuberculosis but also weight loss, prolonged fever and diarrhea. According to the current theory, the HI virus jumped over to humans as a new host around 1930 through multiple zoonoses (currently 13 times) in Central Africa from 3 species of monkeys (it is called SI virus there). According to the same theory, it first showed up around 1980 in the USA in a population of heavily drug-dependent, multiple classically infected homosexual men. This is important, because before that there was no AIDS, only from 1980 on. The around 30 AIDS-defining classic diseases have been around for a long time, but now they are summed up under a new label (AIDS) and are said to be caused by HIV. The *CD4 cell count* is used as a biomarker for the strength of the immune system in the diagnosis. These are cells of the human immune system that are counted and the decreasing number of which is supposed to show the progression of the disease. HIV itself is detected by PCR or antibodies against the HI virus.

So what's the problem?

The problem is that apart from the very sick, severely drug-addicted homosexuals in the United States who really existed, and the diagnostic methods that actually exist, there is no evidence for any of the statements about the origin and effects of HIV.

Most of these people in the US in the 1980s were seriously ill even without a new virus. On the one hand the drugs, in addition to heroin and cocaine in the gay community mainly nitrites (poppers), and on the other hand multiple infections such as syphilis, gonorrhea, hepatitis A and B, herpes, CMV, etc. through frequent unprotected anal intercourse.

- John Lauritsen, Hank Wilson, "*Death Rush: Poppers and AIDS*", 1986  
<http://paganpressbooks.com/jpl/POPPERS.HTM>

*„96-100% of the gay men with AIDS used poppers, usually quite heavily.“*

Cf. on the circumstances at that time,

- Pifer et al., *“Borderline immunodeficiency in male homosexuals: is life-style contributory?”*, South Med J. **1987** Jun;80(6):687-91, 697, <https://www.ncbi.nlm.nih.gov/pubmed/2954211>

*“Results of our study suggest that white Southern male homosexuals without clinical evidence of AIDS who patronize “gay bars” may have significant zinc deficiency and moderately depressed T-helper/T-suppressor cell ratios. No single causative factor could be identified to explain the significantly low zinc and elevated copper levels measured in whole blood, as well as the depressed OKT4/OKT8 cell ratios. **Seventy-four percent of the homosexual male subjects were “recreational” drug abusers, 81% used inhaled nitrites routinely, and 41% routinely treated themselves with antibiotics. Eighty-one percent practiced active and/or passive penile-oral insertion, and 55.5% practiced both active and passive anal intercourse. Of the latter, 19% reported anal bleeding.** Clinically inapparent, though statistically significant, borderline immunodeficiency and aberrant zinc and copper levels may be a consequence of multiple factors comprising the gay bar life-style.”*

However, the toxicity of the alleged antiretroviral therapy (ART, sometimes also referred to as HAART for *highly active antiretroviral therapy*) has been sufficiently proven. Their side effects are indistinguishable from the presumed effects of a virus, as can be read explicitly in the publications.

In the meantime, a positive test for HIV defines the disease. AIDS itself is hardly mentioned any more. HIV is the only disease that knows no spontaneous healing and becomes chronic in all (100%) of the treated cases. In addition, antibodies for this virus only serve to define the disease. According to the current theory, they are otherwise useless. Therefore, *lifelong* therapy is required, according to the, obviously very profitable, theory.

It starts with the fact that nobody knows how the HI virus is supposed to lead to a reduction in CD4 cells and thus, by definition, to AIDS. Cf.

- Coffin, Swanstrom, *“HIV Pathogenesis: Dynamics and Genetics of Viral Populations and Infected Cells”*, Cold Spring Harb Perspect Med. **2013** Jan; 3(1), <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3530041/>

*“HOW DOES HIV-1 CAUSE AIDS? As is apparent from this article and the rest of the collection, **in the 25+ years since its discovery**, we have learned an enormous amount about HIV, **but we still cannot answer the one big question: How does HIV-1 cause AIDS?**”*

*“Even if we knew the mechanism of HIV-mediated cell killing, **we would not know how HIV-1 causes CD4<sup>+</sup> T-cell decline and AIDS in humans.** The observation that virus and cell turnover rates in various SIVs in their natural hosts (such as SIV<sub>sm</sub> in sooty mangabeys), **which do not progress to AIDS**, are essentially identical to those in humans, who do progress, implies that cell killing alone cannot account for AIDS pathogenesis. Indeed, this result is consistent with the **high natural turnover rate of activated effector memory helper T cells, the primary target for HIV-1 infection, on the order of 10<sup>10</sup> cells per day**, of which only a small fraction are infected after the initial primary infection phase.”*

There are far too few cells infected to explain a decrease in CD4 cell counts. Then there is the *bystander cell problem*, especially the non-infected cells die. What sense does that make? Both facts are known to science for more than 25 years.

- Finkel et al. „Apoptosis occurs predominantly in bystander cells and not in productively infected cells of HIV- and SIV-infected lymph nodes.“, Nat Med. **1995** Feb;1(2):129-34,  
<https://www.ncbi.nlm.nih.gov/pubmed/7585008>

*“We show here, using in situ labelling of lymph nodes from HIV-infected children and SIV-infected macaques, that apoptosis occurs **predominantly in bystander cells and not in the productively infected cells themselves.**”*

- Legitimo et al. [in Italian], “Brief analytical review of additional possible mechanisms in the pathogenesis of AIDS”, Pathologica. **1994** Apr;86(2):119-27,  
<https://www.ncbi.nlm.nih.gov/pubmed/7936754>

*“Nonetheless, a number of important issues concerning the pathogenesis of HIV infection remain unresolved. For example, it remains unclear how CD4+ T cells are lost after HIV infection. **The low frequency of infected cells seen even in advanced infection implies that a direct cythopathic effect of HIV on infected CD4+ T cells cannot explain their disappearance.**”*

- Muro-Cacho et al. „Analysis of apoptosis in lymph nodes of HIV-infected persons. Intensity of apoptosis correlates with the general state of activation of the lymphoid tissue and not with stage of disease or viral burden.“, J Immunol May 15, **1995**, 154 (10) 5555-5566;  
<https://www.ncbi.nlm.nih.gov/pubmed/7730654>

*“Taken together, these results indicate that the increased intensity of the apoptotic phenomenon in HIV infection is caused by the general state of immune activation, and **is independent of the progression of HIV disease and of the levels of viral load**”*

- Whitaker, “Re-assessing the virological approach to HIV pathogenesis: can it explain AIDS as an immunological disease?”, J Theor Biol. **1997** Jul 7;187(1):45-56,  
<https://www.ncbi.nlm.nih.gov/pubmed/9236107>

*“However, these attributes - singly and in combination - are shown here to be inadequate to explain the latency, immunological damage, and clinical dynamics of the disease of AIDS. **The virological paradigm cannot explain the disease-free period (clinical latency); the mechanism and dynamics of CD4 T cell loss; the reason for the onset of disease at a given time-point; the relationship of CD4 T cell loss to AIDS-type disease; nor the idiosyncratic constellation of immunological and clinical phenomena that comprise AIDS as a unique syndrome.**”*

- Cloyd et al. "How does HIV cause AIDS? The homing theory.", Mol Med Today. **2000** Mar;6(3):108-11, <https://www.ncbi.nlm.nih.gov/pubmed/10689313>

***"The mechanism by which HIV causes depletion of CD4+ T cells in infected individuals remains unknown. Numerous theories have been proposed, but none can fully explain all of the events observed to occur in patients"***

- Garg, Joshi, „Host and Viral Factors in HIV-Mediated Bystander Apoptosis.", Viruses. **2017** Aug 22;9(8), <https://www.ncbi.nlm.nih.gov/pubmed/28829402>

***"With a limited number of infected cells and vastly disproportionate apoptosis in HIV infected patients, it is believed that apoptosis of uninfected bystander cells plays a significant role in this process."***

***"The number of HIV infected cells in patients is relatively low and cannot solely account for the loss of CD4 cells in vivo. Hence, it is believed that the loss of CD4 cells during HIV infection is due to the process of bystander apoptosis induction."***

***"Apoptosis mediated by HIV infections is more complex than previously thought. A role of both host and viral factors in this phenomenon is becoming increasingly evident."***

One has no idea how the HIV virus is supposed to lead to AIDS. Of course, this opens up space for plenty of research and numerous conjectures. Virologists love to speculate and they are not accountable to anyone. Only one thing is strictly forbidden for them, to question the *HIV=AIDS* dogma.

Hardly anyone knows today that already in 1984 70% of adults with Kaposi's sarcoma, an AIDS-defining cancer, showed no positive test for HIV, cf.

- Gallo et al., "Frequent detection and isolation of cytopathic retroviruses (HTLV-III) from patients with AIDS and at risk for AIDS.", Science. **1984** May 4;224(4648):500-3, <https://www.ncbi.nlm.nih.gov/pubmed/6200936>

*[Table 1. Detection and isolation of HTLV-III from patients with AIDS and pre-AIDS]*

Diagnosis*	Number positive for HTLV-III	Number tested	Percent positive
Pre-AIDS	18	21	85.7
Clinically normal mothers of juvenile AIDS patients	3	4	75.0
Juvenile AIDS	3	8	37.5
Adult AIDS with Kaposi's sarcoma	13	43	30.2
Adult AIDS with opportunistic infections	10	21	47.6
Clinically normal homosexual donors	1	22	4.5
Clinically normal heterosexual donors	0	115	0

\*With the exception of the normal heterosexual donors and some of the clinically normal mothers of juvenile AIDS patients, all others belong to one of the groups of people identified as being at risk for AIDS (homosexual males, intravenous drug users, Haitian immigrants, heterosexual contacts of members of a group at risk, hemophiliacs treated with pooled blood products, recipients of multiple blood transfusions, and infants born of parents belonging to other groups at risk). Pre-AIDS includes patients with unexplained chronic lymphadenopathy and leukopenia, with an inverted T4 (helper)/T8 (suppressor) lymphocyte ratio. The clinically normal, nonpromiscuous, homosexual subjects are from Washington, D.C., and are believed to be at moderate risk. The clinically normal heterosexual donors include both male and female subjects believed not to be at risk for AIDS.

These were the same people whose bodies had been destroyed by years of abuse of nitrates (poppers).

Nobody knows what influences the CD4 cell count. Even sunburn, said to be common in Africa, lowers this biomarker. Also classic infections, such as AIDS-defining tuberculosis have this effect.

- Hersey et al. "Immunological effects of solarium exposure.", Lancet. **1983** Mar 1 2;1(8324):545-8, <https://www.ncbi.nlm.nih.gov/pubmed/6131254>

*"OKT4+ helper T cells were reduced and there was a significant decrease in the OKT4/OKT8 ratio."*

[OKT4+ is an old name for CD4+]

- Skogmar et al., "CD4 Cell Levels during Treatment for Tuberculosis (TB) in Ethiopian Adults and Clinical Markers Associated with CD4 Lymphocytopenia", PLoS One. **2013**; 8(12): e83270, <https://www.ncbi.nlm.nih.gov/pubmed/24358268>

*"In total, 1116 TB patients were included (307 HIV-infected). Among **809 HIV-negative patients, 200 (25%) had subnormal CD4 cell counts (<500 cells/mm<sup>3</sup>)**, with <350 cells/mm<sup>3</sup> in 82 (10%) individuals. **CD4 cell levels increased significantly during the course of ATT in both HIV+ and HIV- TB-patients, but did not reach the levels in healthy subjects.**"*

The recovery of the CD4 cell count after anti-tuberculosis treatment in *HIV-negative* people also shows that tuberculosis influences this bio-marker.

- Luo et al., "Immunological recovery in patients with pulmonary tuberculosis after intensive phase treatment", J Int Med Res. **2018** Sep; 46(9): 3539–3551, <https://www.ncbi.nlm.nih.gov/pubmed/29756540>

*"Inclusion criteria were as follows: ...*

*(4) **seronegative** for human immunodeficiency virus (HIV)"*

*"After 2 months of intensive phase anti-TB treatment, a reduction in the percentage of CD4+ T cells showed a significant restoration similar to that of controls."*

These facts have long been known, cf.

- Jones et al., "CD4 cell counts in human immunodeficiency virus-negative patients with tuberculosis.", Clin Infect Dis. **1997** May;24(5):988-91, <https://www.ncbi.nlm.nih.gov/pubmed/9142808>

*"We evaluated 85 human immunodeficiency virus (HIV)-negative patients with tuberculosis for clinical features and CD4 cell counts. Thirty-seven patients had low CD4 cell counts (mean +/- SD, 341 +/- 116 cells/microL), and 48 patients had normal CD4 cell counts (mean +/- SD, 830 +/- 254 cells/microL)."*

*"The CD4 cell counts returned to normal levels in most patients after 1 month of therapy."*

*"We confirmed previous studies demonstrating that **CD4 cell counts are depressed in HIV-negative patients with tuberculosis**"*

A biomarker "CD4 cell count" makes no sense obviously, especially not in Africa where tuberculosis is death factor number 1. Nobody knows what the normal CD4 cell count in an HIV-negative person is.

- Crampin, „Normal Range of CD4 Cell Counts and Temporal Changes in Two HIV Negative Malawian Populations“, The Open AIDS Journal, **2011**, 5, 74-79, <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3162193/>

*"1.5% and 6% respectively had baseline counts below 350 cells/μl and 1.5% and **2.5% below 250 cells per μl. Transient dips to below 250 cells/μl were observed in seven individuals, with two individuals having persistently low CD4 counts over more than one year.**"*

*„In common with neighbouring countries, **HIV-negative populations in Malawi have CD4 counts considerably lower than European reference ranges, and healthy individuals may have persistently or transiently low counts.** Within Malawi, ranges differ according to the selected population."*

The CD4 cell count also varies with the season, cf.

- Gomo et al., "Predictors and reference values of CD4 and CD8 T lymphocyte counts in pregnancy: a cross sectional study among **HIV negative women in Zimbabwe.**", Cent Afr J Med. **2004** Jan-Feb;50(1-2):10-9, <https://www.ncbi.nlm.nih.gov/pubmed/15490719>

*“The late rainy season was associated with **higher** CD4 counts...”*

*“Gestational age, gravidity, micronutrient status and **season** influence T lymphocyte subset levels and need to be considered when designing clinical management and intervention strategies for pregnant women. The data underscores the need for **local** reference values.”*

These results are important because, on the one hand, the CD4 cell count is now used as a surrogate for an AIDS diagnosis and, on the other hand, this number is used to define so-called *Long Term Non Progressors* (LTNP). These are people who have been measured HIV+, but show no signs of an AIDS-defining disease or a low CD4 cell count even after years.

With a CD4 cell count of 500 cells/ $\mu$ L, the German-Austrian guidelines for antiretroviral therapy of HIV-1 infection recommend starting therapy, even in completely symptom-free people.

- DAIG, „Deutsch-Österreichische Leitlinien zur antiretroviralen Therapie der HIV-1-Infektion“, Version 8 auf der Basis der Konsensuskonferenz vom 10.4.2019, <https://daignet.de/site-content/hiv-therapie/leitlinien-1>

*„**Asymptomatische** Fälle mit CD4+T-Zellen <500/ $\mu$ L: Bei **allen** Patienten mit weniger als 500 CD4-Zellen/ $\mu$ L soll eine Therapie erfolgen. Die Dringlichkeit des Therapiebeginns (binnen Tagen, Wochen oder Monaten) erhöht sich in Abhängigkeit von der CD4+-Zellzahl: Je niedriger die CD4+-Zellzahl, desto dringlicher die Therapie. Bei weniger als 200 CD4+-Zellen steigt das Risiko opportunistischer Folgeerkrankungen erheblich, und Morbidität und Mortalität bleiben trotz erfolgreicher Therapie erhöht (22), der Behandlungsbeginn ist daher dringlich. Bei Vorliegen bestimmter opportunistischer Infektionen sollte die ART wegen des Risikos eines Immunrekonstitutionssyndroms verzögert begonnen werden. Diesbezüglich wird auf die DAIG-Leitlinie Opportunistische Infektionen verwiesen.“*

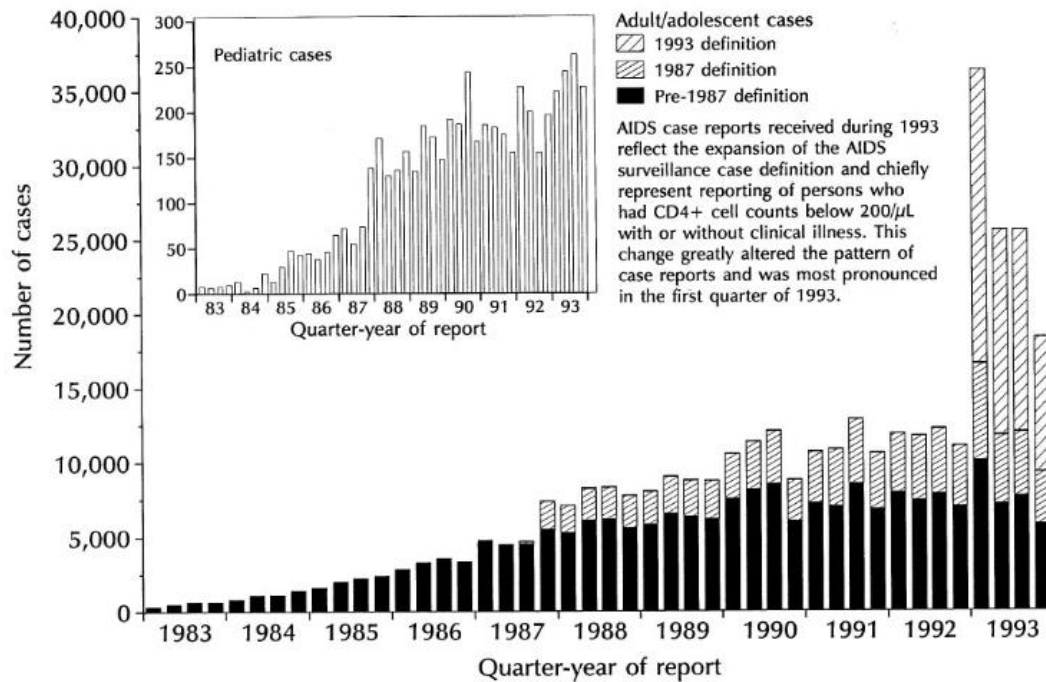
**[Translation: German-Austrian guidelines for antiretroviral therapy of HIV-1 infection, Version 8 based on the consensus conference of April 10, 2019]**

*“**Asymptomatic** cases with CD4 + T cells <500 /  $\mu$ L: Therapy should be given to **all** patients with fewer than 500 CD4 cells/ $\mu$ L. The urgency to start therapy (within days, weeks or months) increases depending on the CD4+ cell count: the lower the CD4+ cell count, the more urgent the therapy. With fewer than 200 CD4+ cells, the risk of opportunistic secondary diseases increases significantly, and morbidity and mortality remain higher despite successful therapy (22), so the start of treatment is urgent. In the presence of certain opportunistic infections, ART should be started with a delay because of the risk of immune reconstitution syndrome. In this regard, reference is made to the DAIG Guideline Opportunistic Infections. ”*

Before “modern medicine” started to use the biomarker “CD4 cell count” to “diagnose” the AID Syndrome, one used a catalog of approx. 30 classic diseases to define the AID syndrome. It should be noted that this catalog was expanded several times to inflate the statistics. Cf.

- CDC, HIV/AIDS Surveillance Report, "U.S. HIV and AIDS cases reported through December 1993", 1993, Year-end Edition, Vol. 5, No. 4, <https://www.cdc.gov/hiv/pdf/library/reports/surveillance/cdc-hiv-surveillance-report-1993-vol-5-4.pdf>

From this document Figure 6 on the *AID syndrome* case numbers (not HIV!). In 1993 the CDC for the last time differentiated the number of cases according to the different definitions of the *AID syndrome*. After that, the *AID syndrome* case numbers were only shown as a uniform, rising (!) curve.



Meanwhile, AIDS-defining diseases are no longer used to diagnose AIDS, but one relies solely on the CD4 cell count. Below about 200 cells/μL one has AIDS, regardless of any symptom. That also works better in the statistics. Any infection can lower the CD4 cell count.

And one sees no connection between the CD4 cell count and the so-called viral load, which is to be determined by quantitative PCR and with which one drives HIV+ measured people into *test madness*.

- Rodriguez et al. „Predictive Value of Plasma HIV RNA Level on Rate of CD4 T-Cell Decline in Untreated HIV Infection”, JAMA, Sep 27, 2006, Vol 296 (12), <https://www.ncbi.nlm.nih.gov/pubmed/17003398>

**„Despite this trend across broad categories of HIV RNA levels, only a small proportion of CD4 cell loss variability (4%-6%) could be explained by presenting plasma HIV RNA level.”**

And what are the consequences of this therapy, which physicians often name "*blessed*" and which is supposed to save people from a slow death? The list is long, cf.

- HIV.gov, “Adverse Effects of Antiretroviral Agents”, Dec. 18, 2019, <https://clinicalinfo.hiv.gov/en/guidelines/adult-and-adolescent-arv/adverse-effects-antiretroviral-agents>

*Bleeding Events*

*Bone Density Effects*

*Bone Marrow Suppression*

*Cardiac Conduction Effects*

*Cardiovascular Disease*

*Cholelithiasis*

*Diabetes Mellitus and Insulin Resistance*

*Dyslipidemia*

*Gastrointestinal Effects*

*Hepatic Effects*

*Hypersensitivity Reaction*

*Excluding rash alone or Stevens-Johnson syndrome*

*Lactic Acidosis*

*Lipodystrophy*

*Myopathy/Elevated Creatine Phosphokinase*

*Nervous System/Psychiatric Effects*

*Rash*

*Renal Effects/Urolithiasis*

*Stevens-Johnson Syndrome/Toxic Epidermal Necrosis*

These are serious and life threatening side effects and that in a lifelong(!) therapy.

This list tends to be too short rather than too long as it does not include some overly toxic substances such as didanosine (ddI), stavudine (d4T), fosamprenavir (FPV), indinavir (IDV), nelfinavir (NFV), saquinavir (SQV) and tipranavir (TPV) which are no longer used. Before that, they had been in use for years, with catastrophic consequences for those treated with them.

What happens is that the presumed therapy damages the human cells. In particular, the nucleoside and nucleotide analogs (so called *nucleoside reverse transcriptase inhibitors*, NRTIs) also contained in the combination therapies damage the mitochondria, i.e. the energy suppliers of cells. This leads to a variety of different tissue damages.

- Gardner, “HIV treatment and associated mitochondrial pathology: review of 25 years of in vitro, animal, and human studies.”, Toxicol Pathol. 2014 Jul;42(5):811-22, <https://www.ncbi.nlm.nih.gov/pubmed/24067671>

*“In 1988, the suggestion that the first antiretroviral drug, zidovudine, was the potential cause of muscle pathology in HIV-infected persons resulted in structural and biochemical patient studies demonstrating acquired mitochondrial dysfunction. Assessment of subsequent nucleoside analog reverse transcriptase inhibitor (NRTI) antiretroviral drugs has indicated that mitochondria are a common target of **NRTI toxicity in multiple tissues**, leading to a wide variety of pathology ranging*

from lipodystrophy to neuropathy. **Overwhelmingly, these complications have emerged during post-licensing human studies.**

**“Millions of patients have been treated with mitochondrially toxic NRTIs and these drugs remain the backbone of antiretroviral rollout in much of sub-Saharan Africa.”**

- Hart et al. “Inflammation-Related Morbidity and Mortality Among HIV-Positive Adults: How Extensive Is It?”, J Acquir Immune Defic Syndr. **2018** Jan 1;77(1):1-7, <https://www.ncbi.nlm.nih.gov/pubmed/28991883>

**“A shift from AIDS-related causes of morbidity and mortality to non-AIDS causes such as *non-AIDS malignancy, liver cirrhosis, end stage renal disease and serious cardiovascular events* occurred in HIV patients nearly one decade ago *due to use of potent antiretroviral therapy.*”**

*“Consistent with two other reports which included participants with lower CD4+ counts, we show that grade 4 events are a major source of morbidity among participants with HIV [26, 27]. Among the participants in our cohort, all of whom had CD4+ counts  $\geq 300$  cells/mm<sup>3</sup> at study entry, the rate of grade 4 events was 3 to 6 times higher than AIDS, CVD (expanded to include less serious events and CVD events that did not meet ERC criteria) or non-AIDS cancer considered separately and was higher than the rate for these three outcomes considered as a single composite outcome.”*

**“Everyone in our investigation was taking suppressive ART. Thus, we can only speculate whether the grade 4 events are due to underlying HIV disease or to ART.”**

As of **2018**. But there is nothing new about these findings. Also the early attempts of a therapy with AZT [zidovudine] were a complete catastrophe.

- Richman et al., “The toxicity of azidothymidine (AZT) in the treatment of patients with AIDS and AIDS-related complex. A double-blind, placebo-controlled trial”, N Engl J Med, **1987** Jul 23;317(4):192-7, <https://pubmed.ncbi.nlm.nih.gov/3299090/>

*“Twenty-one percent of AZT recipients and 4 percent of placebo recipients required multiple red-cell transfusions (P less than 0.001). Neutropenia (less than 500 cells per cubic millimeter) occurred in 16 percent of AZT recipients, as compared with 2 percent of placebo recipients (P less than 0.001).”*

*“Although a subset of patients tolerated AZT for an extended period with few toxic effects, the drug should be administered with caution because of its toxicity and the limited experience with it to date.”*

The trial by Richman et al. (Burroughs & Wellcome) was performed under ridiculously unscientific circumstances. Many placebo participants actually received AZT.

- John Lauritsen, “AZT On Trial”, New York Native (published by Charles Ortleb), 19 October **1987**, <https://www.duesberg.com/articles/jltrial.html>

- Celia Farber, "AIDS and the AZT Scandal: SPIN's 1989 Feature, 'Sins of Omission' - The story of AZT, one of the most toxic, expensive, and controversial drugs in the history of medicine", Nov **1989**, republished Oct 5, 2015, <https://www.spin.com/featured/aids-and-the-azt-scandal-spin-1989-feature-sins-of-omission/>

- Patricia Spitzig, "NDA 19-655 – Site inspection report Massachusetts General Hospital, Boston Mass.", **1987**, <https://archive.org/details/nda-19-665-site-inspection-report-boston-dr.-schooley>

- Patricia Spitzig, "NDA 19-655 – Inspectional observations - Massachusetts General Hospital, Boston Mass.", **1987**, <https://archive.org/details/nda-19-655-report-dr.-spitzig>

If after 44 weeks of therapy 27% of those treated have died, is that no reason to question the therapy, but simply proof of how dangerous the virus is?

- Creagh-Kirk et al., "Survival experience among patients with AIDS receiving zidovudine. Follow-up of patients in a compassionate plea program", JAMA **1988** Nov 25;260(20):3009-15, <https://jamanetwork.com/journals/jama/article-abstract/375200>

*"Through a **compassionate plea program** (Treatment Investigational New Drug), 4805 patients with acquired immunodeficiency syndrome who previously had experienced Pneumocystis carinii pneumonia (PCP) received zidovudine (Retrovir, formerly azidothymidine). **Overall survival at 44 weeks after initiation of therapy was 73% (+/- 2.1%).**"*

And 6 years later,

- Seligmann et al "Concorde: MRC/ANRS randomised double-blind controlled trial of immediate and deferred zidovudine in symptom-free HIV infection" Lancet **1994**; 343: 871-81, <https://www.thelancet.com/journals/lancet/article/PIIS0140-6736%2894%2990006-X/abstract>

***"The results of Concorde do not encourage the early use of zidovudine in symptom-free HIV-infected adults. They also call into question the uncritical use of CD4 cell counts as a surrogate endpoint for assessment of benefit from long-term antiretroviral therapy."***

*"In all, 99 Imm and 38 Def participants **stopped trial capsules because of adverse events**. In only 16 Imm and 2 Def was haematological toxicity the main reason; in the rest it was predominantly gastrointestinal or neurological symptoms (headache) or malaise (table 6). **One or more blood transfusions were received by 18 Imm and 11 Def while they were taking trial capsules.**"*

Some participants of the trial survived only through blood transfusions, as AZT attacks the blood-forming cells in the bone marrow. Compared to the CONCORD trial with a daily dose of 1000 mg AZT, the doses of the alleged therapy have now been reduced dramatically and one has often switched to

less toxic substances than AZT. Lo and behold, people treated in this way live longer. Nevertheless, AZT is still used, e.g. in treating children.

For a while, the diseases caused by the drugs were euphemistically referred to as *HIV-associated* or *HIV-related*. But they do not correspond to the so-called opportunistic infections, i.e. the AIDS-defining diseases. That is why they are now being referred to as *non-HIV co-morbidities*. These are numerous.

- Maggi et al., “Clusterization of co-morbidities and multi-morbidities among persons living with HIV: a cross-sectional study.”, BMC Infect Dis. **2019** Jun 25;19(1):555, <https://www.ncbi.nlm.nih.gov/pubmed/31238916>

“Non-HIV co-morbidities included: **cardiovascular disease, diabetes mellitus, hypertension, oncologic diseases, osteoporosis**, probable case of chronic obstructive pulmonary disease (COPD), hepatitis C virus (HCV) infection, **psychiatric illness, kidney disease.**”

“Table 1 - Characteristics of 1087 patients enrolled in the Cluster Project: **Years since ART initiation 9.0 (4.0–16.0)**”

“The most frequent co-morbidity was **dyslipidemia (55.3%)**, followed by **hypertension (31.4%)**, COPD (29.4%), hepatitis C virus (HCV) infection (25.4, 5.5% with detectable HCVRNA), **psychiatric illness (10.3%)**, diagnosis of **osteopenia/osteoporosis (10.1%)**, **diabetes (6.1%)**, and **renal impairment (4.8%)**; 95 (8.7%) subjects had history of **non-AIDS-defining cancer**. Forty-nine patients (4.5%) had **pCVD events.**”

“Our data evidence that, in spite of mean age lower than 50, **co-morbidity was the rule among our PLWH (82%)**, and that **more than 50% of our patients were multi-morbid**. Moreover, about 30% of them had three or more chronic non-HIV related conditions, thus confirming recent data provided by other studies in the field.”

- Hernández et al., “Increased incidences of noninfectious comorbidities among aging populations living with human immunodeficiency virus in Ecuador: a multicenter retrospective analysis.”, HIV AIDS (Auckl). **2019** Apr 1;11:55-59, <https://www.ncbi.nlm.nih.gov/pubmed/31114389>

“The average age at HIV diagnosis was 34.1 years old and cART in average was started 15.9 months after HIV-diagnosis. **Recruited patients were receiving cART for an average of 59.2±40.2 months. Only 9.9% (n=50) of the patients did not show any NICMs [noninfectious comorbidities]**. Diabetes and pre-diabetes was found in 6% (n=30) and 16.3% (n=82) patients, respectively; however, dyslipidemia and overweight/obesity was frequent, as they affected 41.4% (n=208) and 36.4% (n=183) patients, respectively.”

“Conclusion: **Prevalence of NICMs among subjects under cART was greater than that reported among the Ecuadorian general population**, therefore specific public health actions are required to make patients aware of and prevent NICMs among PLHIV in Ecuador.”

The non-HIV co-morbidities correspond 1:1 to the side effects of the alleged therapy.

In the press the virologists speculate on these serious and ultimately fatal comorbidities. As a virologist you are not accountable to anyone.

- Pamela Dörhöfer, „HIV und Aids: Kampf gegen die Stigmatisierung – Interview mit Jürgen Rockstroh, Präsident der Europäischen HIV/Aids-Gesellschaft“, Frankfurter Rundschau, 19 Nov **2019**, <https://www.fr.de/wissen/hiv-aids-kampf-gegen-stigmatisierung-13201336.html>

**„Liegen bereits Erkenntnisse vor, ob eine langjährige Infektion und Einnahme der Tabletten verstärkt zu bestimmten Begleiterkrankungen führen?“**

*Es gibt verschiedene Forschungsprojekte, die sich mit dieser Frage beschäftigen. Wir wissen, dass **Bluthochdruck, Diabetes Mellitus und Osteoporose** häufiger und bereits in jüngerem Lebensalter auftreten. Das hängt **vermutlich** damit zusammen, dass bei Infizierten – auch wenn sie mit Medikamenten die Viruslast gering halten – das Immunsystem ständig stimuliert wird. Das löst eine Entzündungsreaktion aus, die all diese Erkrankungen begünstigt.“*

**[Translation: HIV and AIDS: Fighting stigmatization - Interview with Jürgen Rockstroh, President of the European HIV/AIDS Society]**

**“Are there any findings as to whether a long-term infection and taking the tablets lead additionally to certain concomitant diseases?”**

*There are various research projects dealing with this question. We know that **high blood pressure, diabetes mellitus and osteoporosis** occur more frequently and at a younger age. This is **presumably** due to the fact that in infected people - even if they keep the viral load low with medication - the immune system is constantly stimulated. It triggers an inflammatory reaction that favors all these diseases.”*

The drugs supposedly work, but an adhoc assumed increased level of inflammation leads to diseases that correspond 1:1 to the side effects of the alleged drugs? This is the case in the interviews with the Frankfurter Rundschau.

The fatal consequences of antiretroviral therapy are most evident in misdiagnosed *HIV-negative* people, cf.

- Julian Guthrie, SF Chronicle, “HAYWARD / False diagnosis of HIV discovered after 8 years / Veteran's life severely affected after VA doctor made mistake” ,August 28, **2004**, <https://www.sfgate.com/health/article/HAYWARD-False-diagnosis-of-HIV-discovered-after-2729917.php>

*“Earlier this month, Malone, 59, was summoned to his doctor's office. He listened as the doctor delivered the stunning news: **He is HIV negative.**”*

*"An HIV-positive person can have good T-cell counts and undetectable viral loads over a long period of time," Pridmore said. "And in this case, the patient exhibited symptoms that could be consistent with an HIV diagnosis."*

*„In a September 2003 letter from Karp, Malone was classified as "permanently disabled and unable to work or participate in any stressful situation whatsoever." His medical prognosis was deemed "very poor." The letter said **Malone was being treated for 20 medical conditions, the first condition being HIV.** The sixth item on the list, nausea and vomiting, was said to be "**related to condition 1**"."*

*„Malone, who is thin and voluble and walks with a cane, said that **he attributed his frequent nausea, vomiting, diarrhea and weight loss to being HIV-positive.**"*

The assumptions to which Dr. Rockstroh refers in the interview with the Frankfurt Rundschau are deadly nonsense. Just like the *immune reconstitution syndrome*, also adhoc assumed by medicine, which can hardly be surpassed in terms of patient contempt. The treated people die precisely because the therapy is so successful. Cf.

- Colomba und Rubino, *"The Downside of an Effective cART: The Immune Restoration Disease"*, Current Perspectives in HIV Infection, Ed. Shailendra K. Saxena, April 10, **2013**, <https://www.intechopen.com/books/current-perspectives-in-hiv-infection/the-downside-of-an-effective-cart-the-immune-restoration-disease>

*"This phenomenon is known as a multitude of names including "immune reconstitution inflammatory syndrome (IRIS)", "immune reconstitution or restoration disease" (IRD) or immune reconstitution syndrome" and includes various forms of a clinical deterioration as a consequence of a rapid and dysregulated restoration of antigen specific immune responses causing an exuberant inflammatory reaction and a cytokines storm. This was first noted following the introduction of zidovudine monotherapy in the early 1990s, [...]."*

The patients' health becomes worse or they die because the immune response, which, thanks to the therapy, is supposed to overshoot, according to this very convenient theory. Given the many and severe side effects of the medication and the fact that it is not even clear how the HI virus is supposed to lead to a deterioration in the immune system, see above, this is remarkably thin.

But if you question that, you don't risk your scientific career, it will then be over. There is a reason for that. The science of HIV and AIDS consists of little more than guesswork.

Very few HIV+ measured people in therapy die from the diseases from the AIDS catalog, cf.

- Lifson et al, *"Determination of the underlying cause of death in three multicenter international HIV clinical trials."*, HIV Clin Trials. **2008** May-Jun;9(3):177-85, <https://www.ncbi.nlm.nih.gov/pubmed/18547904>

*"Of 453 deaths reported through January 14, 2008, underlying causes were as follows: **10% AIDS-defining diseases**, 21% non-AIDS malignancies, 9% cardiac diseases, 9% liver disease, 8% non-AIDS-defining infections, 5% suicides, 5% other traumatic events/accidents, 4% drug overdoses/acute intoxications, 11% other causes, and 18% unknown."*

That too has been known for years. But nobody looks at the consequences of the alleged therapy.

In spite of all this nobody questions the hypothesis of the lentivirus, which is said to lead to a breakdown of the immune system after 10-15 years, cf.

- Fauci et al. "Immunopathogenic Mechanisms of HIV Infection", Ann Intern Med. **1996**; 124(7), p. 654-663, <http://annals.org/aim/fullarticle/709558/immunopathogenic-mechanisms-hiv-infection>

*"The duration of clinical latency varies, but progression to the acquired immunodeficiency syndrome typically occurs **after a mean of approximately 10 years**."*

Unless you are a *Long-Term-Non-Progressor* (LTNP). Then it can take as long as you like. This has long been known, cf.

- Hoover et al., "Long-term survival without clinical AIDS after CD4+ cell counts fall below 200 x 10(6)/l.", AIDS. **1995** Feb;9(2):145-52, <https://www.ncbi.nlm.nih.gov/pubmed/7718184>  
*"Although antiretroviral therapy and Pneumocystis carinii prophylaxis extend AIDS-free survival, **45% of the group who were AIDS-free > or = 3 years after CD4+ cells fell below 200 x 10(6)/l had not used these treatments**."*

*"CONCLUSIONS:*

***Significant numbers of individuals remain free of illnesses and AIDS symptoms > or = 3 years after CD4+ cell counts drop below 200 x 10(6)/l. This occurs even in the absence of treatment.** The associations seen here suggest that host and viral factors play important roles."*

This result from 1995 would no longer be valid today. As stated above, the CD4 cell count, unusable as it is, has replaced the AIDS diagnosis according to the catalog diseases. By today's definition, due to their CD4 cell count these people would not have been counted as AIDS-free, though they showed no symptoms. In therapy they will develop symptoms.

Depending on the publication, the prevalence of LTNP (*Long Term Non Progressors*), i.e. HIV+ people who show no signs of AIDS, amount to up to 22%. The values fluctuate depending on the study, cf.

- Sabin, Lundgren, "The natural history of HIV infection", Current Opinion in HIV and AIDS: July **2013**, Vol 8(4), p. 311–317, [https://journals.lww.com/co-hivandaids/fulltext/2013/07000/The\\_natural\\_history\\_of\\_HIV\\_infection.10.aspx](https://journals.lww.com/co-hivandaids/fulltext/2013/07000/The_natural_history_of_HIV_infection.10.aspx)

[Table 1]

Table 1. Definitions of long-term nonprogressors used in recent studies						
Author (reference)	Symptoms allowed	ART allowed	Period of follow-up	CD4 requirement	Additional requirements/comments	Reported prevalence
Madec <i>et al.</i> [3]	Asymptomatic	No ART	>8 years after first positive HIV test	All $\geq 500$ cells/ $\mu$ l	Study includes a high proportion of known seroconverters	9.0%
Okulicz <i>et al.</i> [4]	No AIDS	No ART	>7 years after diagnosis	All $\geq 500$ cells/ $\mu$ l	–	5.0%
	No AIDS	No ART	>10 years after diagnosis	All $\geq 500$ cells/ $\mu$ l	–	2.0%
Grabar <i>et al.</i> [5]	Asymptomatic	No ART	>8 years after diagnosis	Nadir $>500$ cells/ $\mu$ l	At least three CD4 and HIV RNA assessments available in 5 years prior to 2005	22.3%
	Asymptomatic	No ART	>8 years after diagnosis	Nadir $>600$ cells/ $\mu$ l	As above	11.4%
	Asymptomatic	No ART	>8 years after diagnosis	Nadir $>600$ cells/ $\mu$ l	As above, and positive CD4 slope over 5 years prior to 2005	2.8%
Mandalia <i>et al.</i> [6**]	Asymptomatic	No ART	>7 years after diagnosis	$>450$ cells/ $\mu$ l	Stable CD4 slope ( $\geq 0$ cells/ $\mu$ l per year) over entire follow-up period	0.2%
Gaardbo <i>et al.</i> [7]	Not stated	No ART	>10 years after diagnosis	$>350$ cells/ $\mu$ l	Viral load $>5000$ copies/ml	N = 14, prevalence not stated
Ballana <i>et al.</i> [8]	Not stated	No ART	>10 years after diagnosis	All $>500$ cells/ $\mu$ l	Viral load $<10000$ copies/ml	N = 155, prevalence not stated

ART, antiretroviral therapy.

But, the criteria for LTNP are partly designed in such a way that even the healthiest person cannot meet them: the slope of the CD4 curve (“stable CD4 slope”) must never be negative (always  $\geq 0$ ). I.e. a flu-like infection on the examination, which leads to a temporary drop in the CD4 number, and no LTNP anymore.

The number of LTNPs is calculated in a way to be artificially small. This is important because HIV+ without AIDS in 22% and more of those affected means a blatant violation of Koch’s postulates.

Nobody questions the hypothesis of the currently assumed 13 different zoonoses from at least 3 species of monkeys, gorillas (SIVgor), chimpanzees (SIVchz) and Shooty Mangabeys (SIVsm) to humans, around 1930 in Africa, which is supposed to have led to the development of the HI virus, cf.

- Hahn et al. “AIDS as a zoonosis: scientific and public health implications.”, Science. **2000** Jan 28; 287(5453):607-14, <https://www.ncbi.nlm.nih.gov/pubmed/10649986>

“Evidence of simian immunodeficiency virus (SIV) infection has been reported for 26 different species of African nonhuman primates. Two of these viruses, SIVcpz from **chimpanzees** and SIVsm from **sooty mangabeys**, are the cause of acquired immunodeficiency syndrome (AIDS) in humans. Together, they have been transmitted to humans **on at least seven occasions**.”

“How the AIDS epidemic actually began, what the contributing factors were, and **why it appeared in the mid- to late 20th century (and not before) are not known**. Whatever the final answers are, they must account for

- at least seven separate introductions of SIVcpz and SIVsm viruses into humans;
- the fact that the HIV-1 group M, N, and O viruses are significantly more closely related to SIVcpz viruses from *P. t. troglodytes* than to the single SIVcpz isolate from *P. t. schweinfurthii*; and
- the estimation of 1930 (range 1910 to 1950) as the timing of the last common ancestor of the HIV-1 group M viruses.”

Earlier than 1910 is not possible, otherwise there should have been an epidemic earlier. Later than 1950 does not work due to the >10 year latency of the alleged slow virus and the first cases in 1981 in the USA. More recent publications now speak of at least 13x transitions between the species, cf.

- Peeters et al., „Origin and diversity of human retroviruses.”, AIDS Rev. **2014** Jan-Mar;16(1):23-34, <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4289907/>

*“More in detailed studies showed that SIVs from chimpanzees and gorillas have crossed the species barrier on at least **four** occasions leading to HIV-1 group M, N, O and P in humans [6,23]. The different HIV-2 groups are the result from at least **nine** independant transmissions of SIVs from sooty mangabeys in west Africa [6,23,24].”*

And the number of zoonoses will probably grow, cf. ibid,

***“Already 13 transmissions involving 3 different NHP species to humans have been documented, 4 for HIV-1 and 9 for HIV-2. Most likely other cross-species occurred in the past but remained undetected, because the virus could not adapt to his new host or was not introduced into an environment where conditions for efficient and rapid spread were present. Today humans are still exposed to a wide diversity of SIVs through hunting and butchering NHPs for bushmeat.”***

Nobody asks why these alleged zoonoses did not happen much earlier when humans and their ancestors lived under much worse hygienic conditions than today.

According to theory, 2 different putatively pathogenic HI virus groups arose at the same time, HIV-1 and HIV-2, which differ in their genome by > 45%, cf.

- Motomura et al., “Genetic Recombination between Human Immunodeficiency Virus Type 1 (HIV-1) and HIV-2, Two Distinct Human Lentiviruses”, J Virol. **2008** Feb; 82(4): 1923–1933, <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2258735/>

*“HIV-1 and HIV-2 have similar genetic structures; however, they exhibit significant sequence variation. For example, **the two virus strains used in this study contain only 55% nucleotide sequence identity** in the viral genome and 54%, 55%, and 35% amino acid sequence identity in gag, pol, and env, respectively.”*

This is a very bizarre coincidence. And there would be 2 remarkably different *human immunodeficiency viruses*.

What is the point of phylogenetic analysis of the family tree of the HI virus if no two HIV+ people on this planet carry the same virus? On this point, we can refer to none other than the co-discoverer of the HI Virus and Nobel Prize winner Françoise Barré-Sinoussi, cf.

- Barré-Sinoussi et al., “Expert consensus statement on the science of HIV in the context of criminal law.”, J Int AIDS Soc. **2018** Jul;21(7):e25161, <https://www.ncbi.nlm.nih.gov/pubmed/30044059>

*“Mutations of the virus occur repeatedly so that every person living with HIV has more than one virus variant [154]. During transmission, a limited number of virus variants (one to a few) are transmitted, but these will also mutate to form new variants **so that no two persons’ HIV is identical** [155].”*

The alleged evidence of a (or at least 13) zoonosis (zoonoses) in Africa around 1930 is simply nonsense. The *molecular clock analysis* method leads to the conclusion that SI viruses originated in monkeys about 500 years ago. Cf.

- Wertheim, Worobey, “Dating the age of the SIV lineages that gave rise to HIV-1 and HIV-2.”, PLoS Comput Biol. **2009** May;5(5):e1000377, <https://www.ncbi.nlm.nih.gov/pubmed/19412344>

*“Here, we use **relaxed molecular clock** dating techniques to estimate the time of most recent common ancestor for the SIVs infecting chimpanzees and sooty mangabeys, the reservoirs of HIV-1 and HIV-2, respectively. The date of the most recent common ancestor of SIV in chimpanzees is estimated to be **1492** (1266-1685), and the date in sooty mangabeys is estimated to be **1809** (1729-1875).”*

*“Comparisons between the SIV most recent common ancestor dates and those of the HIV lineages suggest a **difference on the order of only hundreds of years**. Our results suggest either that SIV is a surprisingly young lentiviral lineage or that SIV and, perhaps, HIV dating estimates are seriously compromised by unaccounted-for biases.”*

This method makes obviously no sense. Given the prevalence of lentiviruses in the animal kingdom, that's not even close to plausible. On the contrary, it can be assumed that SIV, like HIV, is several million years old.

- Compton and Emerman, “Convergence and Divergence in the Evolution of the APOBEC3G-Vif Interaction Reveal Ancient Origins of Simian Immunodeficiency Viruses”, PLoS Pathog 9(1): e1003135, Jan 24, **2013**, <https://journals.plos.org/plospathogens/article?id=10.1371/journal.ppat.1003135>

*“The pattern of adaptive mutation suggests that SIV has been infecting OWM **on timescale of millions of years**.”*

But AIDS has only existed since the early 1980s. The first 5 cases were reported in 1981, cf.

- Gottlieb et al., “Pneumocystis Pneumonia - Los Angeles”, Morbidity and mortality weekly report, Vol. 30, no. 21, June 5, **1981**, <https://stacks.cdc.gov/view/cdc/1261>

And until today, HIV/AIDS in industrialized countries is limited to so-called risk groups (mostly MSM, men-having-sex-with-men). A heterosexual epidemic does not take place to this day. Why should it be any different in Africa? In Africa there is hunger, misery, contaminated water, heavy metals and many other things. None of this matters any more once a person's is measured HIV+. Former South African President Thabo Mbeki once put it this way,

- Thabo Mbeki, Mail & Guardian, “Mbeki addresses 'Aids denialism' criticism”, 07 Mar 2016, <https://mg.co.za/article/2016-03-07-a-brief-commentary-on-the-question-of-hiv-and-aids>

*“The question that arises from this is – why! **Why does the same Virus behave differently in the US and Western Europe from the way it behaves in Southern Africa!**”*

*“**Why did it come about that so much noise was made internationally about the 9th leading cause of death in our country, with not even so much as a whimper about the 1st leading cause of death, tuberculosis?***

*Why would the South African Government, knowing the health condition of its own population very well, have been expected so to focus on the 9th leading cause of death as virtually to treat as less urgent and important the first eight (8) leading causes of death, even taken together? Did this have to do with the fact that South Africa could be a lucrative market for the sale of ARVs, as it now is?”*

*“**Poverty is the main reason why babies are not vaccinated, why clean water and sanitation are not provided, why curative drugs and other treatments are unavailable and why mothers die in childbirth.** It is the underlying cause of reduced life expectancy, handicap, disability and starvation. Poverty is a major contributor to mental illness, stress, suicide, family disintegration and substance abuse. Every year in the developing world 12.2 million children under 5 years die, most of them from causes which could be prevented for just a few US cents per child. They die largely because of world indifference, but most of all they die because they are poor.”*

Mr. Mbeki and many others have been severely attacked for this position. It goes as far as the absurd accusation that they failed to save human lives because they did not introduce the antiretroviral therapy based on the virus hypothesis of AIDS quickly enough.

We have seen the results of this alleged therapy above. It was only after the doses were greatly reduced that people lived longer.

One has tried to underpin the attacks against the critics with crude model calculations. Cf. for example,

- Chigwedere et al., “Estimating the lost benefits of antiretroviral drug use in South Africa.”, J Acquir Immune Defic Syndr. **2008** Dec 1;49(4):410-5, <https://www.ncbi.nlm.nih.gov/pubmed/19186354>

*“To estimate the lost benefits of ARV drug use in South Africa, we compared the actual number of persons who received ARVs for treatment or PMTCT between 2000 and 2005 with what was*

*reasonably feasible in the country during that period. **The difference, multiplied by the average efficacy of ARV treatment or PMTCT prophylaxis gives us the lost benefits of ARV use.***"

The problem is that there is no evidence for the presumed effectiveness of HAART as a lifelong(!) therapy, on the contrary. I.e. the multiplicative factor of the mean AVR efficacy (... *average efficacy of ARV treatment* ...) is equal to zero. These drugs do not save lives. The lower the doses, the more slowly they kill.

Thousands of times this model nonsense has been adopted and spread in the media. And the journalists stood there with their breast swollen with pride and patted themselves on the shoulders, what good people they are. Once again the planet was saved.

We could only touch on a few topics here. We did not talk about the still missing animal model of AIDS (*apes don't get AIDS*), the immunosuppressive effects of drugs, malnutrition or heavy metals, the lack of any evidence for the *slow virus* theory, the multiple forms of alleged therapeutic failure (including so-called PCR blips) with which one would like to conceal the ineffectiveness of the therapy, the connection between ART and non-AIDS-defining cancer, the complexity that arises from endogenous retroviruses in the human genome (HERV) for detection, the nonsense of pre-exposure prophylaxis (PrEP) where healthy people are treated with toxic agents and get sick, the billions in profits thanks to the virus hypothesis of AIDS, or the statistical tricks with which one has inflated the number of cases, for example by expanding the list of AIDS-defining diseases several times, or asking why the population in Africa continues to grow rapidly despite a deadly virus. Nor have we talked about the disastrous consequences of the therapy in pregnant women in Africa, for both mother and fetus.

Of course, like the antibody tests, the HIV PCR tests are a disaster. For HIV, too, PCR is its own gold standard. Only representatively here,

- Jenn Morson, „*How I Tested HIV Positive — Nine Times — While Pregnant*“, May 25 2016, <https://www.ozy.com/true-story/how-i-tested-hiv-positive-nine-times-while-pregnant/68859>

Molecular biology. The science of life. And has one made of it? A profit center - above a morgue.

Everyone should have understood by now why virology in the *corona (lockdown) crisis* persists in its positions against all evidence. Several million people who have been treated to death prevent any deviation from the zoonosis nonsense and the belief in the "*blessed therapy*".